Allergy Abstracts

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Dermatology

Some Endocrine Aspects of Skin Sensitization and Primary Irritation. II. Observations on the Influence of Adrenalectomy on Cutaneous Irritation and Sensitization Induced by Various Chemical Compounds. Nilzen, A.: J. Invest. Dermat. **19**: 337, 1952.

Guinea pigs were adrenalectomized either before or after being sensitized with 2:4-dinitrochlorobenzene. Control animals were similarly sensitized, but were sham operated. Following adrenalectomy, both specific (2:4 dinitrochloro-benzene) and nonspecific (croton oil) skin responses were enhanced. This was demonstrated by the fact that weaker testing solutions were capable of inducing specific and nonspecific reactions. It was found that less time was required for inducing sensitivity. Plasma from adrenalectomized animals seemed to transfer skin reactivity more readily than the plasma from control animals. Whether this was due to the influence of specific or nonspecific factors was not clear. It was not possible to induce cutaneous sensitivity in either normal or adrenalectomized mice. The skin reaction to primary irritants on mice was not enhanced by adrenalectomy. The author suggests that the enhanced reactions seen after adrenalectomy are due to a weakened topical defense mechanism caused by a lack of adrenal cortical hormones.

R. W.

Eczematous Contact Dermatitis of the Palm Due to Toothpaste. Loewenthal, H.: New York State J. Med. **52**: 1437, 1952.

A 46-year-old woman complained of repeated attacks of itching, swelling, and blister formation of the left hand for the last six months. The swelling and itching would subside and recur at about weekly intervals. The lesions were confined to the proximal part of the hypothenar eminence, the thenar eminence, the web between thumb and index finger, and the interdigital webs of the left hand. After a long search, toothpaste was found to be the offending agent. The patient was edentulous and had worn full upper and lower dentures for a number of years. At least once a week, she cleaned them thoroughly by gripping them in the palm of her left hand and scrubbing them with toothpaste and a toothbrush. The patient had been using "Amm-i-dent" toothpaste for about six months before the dermatitis began. The dermatitis became worse several hours after the toothpaste was used. After discontinuation of the use of this brand of toothpaste and the institution of local treatment with zinc oxide ointment, the dermatitis cleared in about eight weeks. A positive patch reaction was obtained with "Amm-i-dent" toothpaste. The "baxin" component of the toothpaste was thought to be responsible for the dermatitis in this case. B. S.

Treatment of Various Dermatoses With Topical Application of Panthenol. Kline, P. R., and Caldwell, A.: New York State J. Med. **52**: 1141, 1952.

The therapeutic value of Panthenol, the active alcohol analog of pantothenic acid, was studied in 31 patients with various types of chronic dermatoses. Two to 5 per cent Panthenol cream was applied topically. Excellent results were obtained in 21 of the 31 cases. The Panthenol cream showed clinical evidence of epithelizing stimulation. It also had an antipruritic and an antibacterial effect in these dermatoses. There was no evidence of sensitization to Panthenol or to the vehicle. B. S.

Sensitivity to Ointments Containing Wool Fat. Schwarzfeld, H. K.: U. S. Armed Forces M. J. **3**: 1371, 1952.

Aquaphor contains 6 parts of the fatty acids and sterols derived from lanolin and 94 parts of petroleum hydrocarbons. Ten patients with contact dermatitis were patch tested with lanolin and Aquaphor. Of these, 2 were sensitive to lanolin and Aquaphor, 4 were sensitive to lanolin alone, and 4 were sensitive to Aquaphor alone. Four of these patients were further patch tested with cholesterol crystals, cholesterol in 10 per cent mineral oil, lipids derived from lanolin and used in Aquaphor, 35 per cent cholesterol and oxycholesterins from lanolin in peanut oil, and petrolatum. In 3 of these patients, only the lanolin lipids used to make Aquaphor gave positive reactions. Cholesterol was not the sensitizer in these 4 patients. Hence, in this series, only 2 patients were sensitive to a factor common to both lanolin and Aquaphor. Among the other 8 patients, the sensitizer was distinct and different in each half of the group. E. W.

Drug Eruption From Paludrine (Proguanil) Hydrochloride: Report of a Case. Lowenthal, L. J. A.: Brit. J. Dermat. **64**: 106, 1952.

Hypersensitivity to Paludrine, a drug used in the treatment and prevention of malaria, was encountered in a 48-year-old white South African man.

This patient had 3 attacks manifested by paresthesia, edema, erythema, and desquamation of the hands and feet, as well as furrowing of the nails. Each attack followed the use of Paludrine for the prevention of malaria. When the drug was discontinued the lesions cleared. E. W.

Exacerbation of Ivy Dermatitis by Rhus Antigen Injections. Reyer, W. A.: Ann. Allergy **11**: 91, 1953.

Following treatment with Rhus antigen, 4 patients with poison ivy dermatitis developed a flare-up of the dermatitis which progressed to exfoliation. F.

Delayed Cutaneous Hypersensitivity in Sarcoidosis. Friou, G. J.: J. Clin. Investigation **31**: 630, 1952.

Twenty-two patients with proved sarcoidosis and 64 controls were tested cutaneously with tuberculin, oidiomycin, trichophytin, and mumps antigen. The sarcoidosis patients were less reactive to all of the antigens tested than were the control individuals. The writer concluded that the "decrease in delayed hypersensitive reactions in sarcoidosis is not limited to tuberculin, but is a broad, immunologically nonspecific phenomenon." K.

Absorption of Pyribenzamine Through Intact and Damaged Skin. Michelfelder, T. J., and Peck, S. M.: J. Invest. Dermat. **19**: 237, 1952.

Two, five, ten, and twenty per cent concentrations of pyribenzamine (PBZ) hydrochloride and PBZ base were incorporated into the following bases: cream, ointment, carbowax base, and vanishing cream. Pyribenzamine excretion levels in the urine were measured by the Perlman test. Efficacy of the absorption of PBZ from the various vehicles was measured by the effect produced on histamine wheals. Individuals with normal skin and subjects with various skin disorders were used for the testing. Three grams of the ointment being tested was rubbed for ten minutes into a site 10 by 15 cm. on the flexor surface of the forearm. Five minutes later 0.05 ml. of histamine was injected intracutaneously into the cleansed area. Control injections of histamine were always made simultaneously on the opposite forearm on an unprepared site. When concentrations less than 10 per cent were used, the effect was so slight that the various vehicles could not be adequately evaluated. When a 10 per cent concentration of PBZ hydrochloride was applied to normal skin regardless of the type of vehicle employed, it was barely detectable in the urine. PBZ hydrochloride was demonstrable in the urine after inunction to damaged skin from all four vehicles. PBZ base was absorbed through damaged skin more readily than PBZ hydrochloride. The type and degree of damage to the skin were more important than the vehicle in influencing the absorption of both PBZ hydrochloride and PBZ base from diseased skin. The maximum inhibitory effect upon histamine whealing from PBZ hydrochloride occurred ninety minutes following its application and the effect persisted for about twenty-four hours. PBZ base had about the same total antiwhealing effect but its action was sustained twice as long. On the whole, the longer the inunction and the greater the concentration of the drug, the larger the quantity of drug absorbed from any one of the vehicles. PBZ hydrochloride penetrated normal skin most easily in a water-soluble base whereas PBZ base was best absorbed in normal skin from a fatty base. PBZ base was better absorbed than PBZ hydrochloride even when the latter was incorporated in a water-soluble vehicle. It is suggested that "the property of being lipoid-soluble is more important than the vehicle itself." R. W.

Sodium Para-Aminobenzoate ("PABA") in the Treatment of Dermatoses. Grayson, L. D., and Steiner, K.: J. Invest. Dermat. **19**: 463, 1952.

Twenty dermatologic patients, 8 of whom had atopic dermatitis, were given "PABA" orally for periods ranging from two weeks to several months. The usual dose was 16 Gm. daily given in 4 equally divided amounts. Only 2 of the 8 patients with atopic dermatitis had good results. The effect was only temporary in one of these cases. Where any improvement occurred, it was most noticeable in these individuals with some form of allergic dermatitis. After a few days of therapy, there occurred a diminution or disappearance of edema and a fading of the inflammatory process. Pruritus usually improved quickly but often temporarily increased at the end of the first week of treatment. Side reactions from the drug were minimal. Reaginic reactions were not affected by "PABA" and patients known to be allergic to various foods still had exacerbations of their dermatitis following ingestion of the specific allergen even while on the drug. Thorne test reactions showed comparable diminution in total eosinophil counts after both "PABA" and ACTH.

It is suggested that "PABA" may be helpful in cases of therapy-resistant inflammatory dermatoses and that its use by the parenteral route should be further investigated. R. W.

Industrial Dermatitis Due to Contact With Brass. Morris, G. E.: New England J. Med. **246**: 366, 1952.

Five cases of contact dermatitis due to brass are reported. Each patient had a job which entailed the handling of brass parts. Patch tests performed with brass were positive in all cases. In one patient, positive patch tests reactions were also obtained with pure copper and zinc, which are the most important elements in brass alloy. Two other patients demonstrated positive reactions to copper, but negative reactions to zinc. This is the first report of dermatitis from contact with brass. B. S.

Eczema and Dermatitis. Effects of Sodium Para-Aminobenzoate. Loewenthal, L. J. A.: South African M. J. **26**: 661, 1952.

The suppressive action of sodium para-aminobenzoate upon eczema and dermatitis was studied in 200 cases. The dosage varied from 6 to 12 Gm. daily, administered orally in 4 divided doses for adults and proportionately less for children. The earliest sign of improvement was relief of symptoms, occasionally with visible regression of the rash.

Of the 200 patients treated, 41 were improved after one day of treatment, 46 after two days, and 78 after one week of treatment. A total of 165 patients (82.5 per cent) improved; 35 showed no improvement. The best results were observed in atopic dermatitis and chronic disseminated neurodermatitis. Prolonged treatment was carried out in 57 cases for periods of from one to six months. Forty of these cases showed sustained relief.

Toxic effects of the drug included transient nausea, faintness, heartburn, abdominal cramps. Septic complications including local impetiginization, furunculosis, and tonsillitis developed in 27 cases, and were controlled by penicillin. The growth-promoting effect of this drug on bacteria is notable. H.

Glomerulonephritis Associated With Atopic Dermatitis. Steiner, K.: New England J. Med. **247**: 201, 1952.

Among 41 patients with atopic dermatitis, 7 were found to have glomerulonephritis. In 4 of these cases, the course of the 2 diseases paralleled each

other. In 11 other cases, minimal urinary abnormalities were found, but the diagnosis of glomerulonephritis could not be made. Among a control group of 20 patients with nonatopic skin diseases, no urinary abnormalities were found on repeated examinations. The nephritis in the 7 cases could not be correlated with the use of potentially nephrotoxic substances such as tars. In one case, a streptococcal pharyngeal infection was present, but both the nephritis and the dermatitis subsided while the antistreptolysin titers were still rising. The author postulates that the glomerulonephritis in these cases is a manifestation of generalized atopic hypersensitiveness. B. S.

A Study of the Local Tissue Reactions in Man to Cortisone and Compound F. VI. Histopathological Studies of the Local Effect of Compound F in Normal and Pathologic Skin of Man. Goldman, L., O'Hara, H., and Baskett, J.: J. Invest. Dermat. **20**: 271, 1953.

The skins of patients with various dermatologic disorders and those of normal control subjects were injected locally with compound F, cortisone acetate, or cortisone tricarballylate. Skin biopsy specimens were examined to determine what changes occurred microscopically in the normal and pathologic skin as a result of the introduction of these hormones. The responses observed in normal skin are fully described. A definite inhibition of the inflammatory reaction was demonstrated in eczema and neurodermatitis as well as in tuberculin, sarcoid, and lymphomatous reactions. Significant changes were not noted in histamine wheals or urticarial reactions. It is suggested that further study with this technique may help to uncover many unknown facts about dermatologic diseases. R. W.

Local Tissue Reactions to Cortisone and Hydrocortisone (Compound F) in Man. VIII. Studies of Local Therapeutic Effect of Hydrocortisone in Diseases of the Skin. Goldman, L., and Preston, R. H.: Arch. Dermat. & Syph. **67**: 163, 1953.

The local therapeutic effects of Compound F were studied over a two-year period in 460 patients with a wide variety of dermatoses. Compound F was administered by syringe and needle, by the hypospray jet injection apparatus, in various ointment vehicles, and as a suspension in various solutions.

In ointment form, Compound F was ineffective. As wet dressings, results with Compound F were also disappointing except in cases of dermatitis of the eyelids. The best results were obtained with the local injection of Compound F acetate directly into the skin lesions. There was no benefit noted in 28 cases of urticaria, histamine wheals, and dermographism. C.

Passive Transfer of Tuberculin Sensitivity to Patients With Sarcoidosis. Urbach, F., Sones, M., and Israel, H. L.: New England J. Med. **247**: 794, 1952.

Skin sites on tuberculin-negative subjects who did not have sarcoidosis were sensitized with washed white blood cells obtained from tuberculin-positive donors and tuberculin-negative donors with and without sarcoidosis. Eighteen hours after inoculation, the sensitized sites were tested with 0.005 mg. of PPD, and reactions were read forty-eight hours later. The tuberculin sensitivity was passively transferred to the tuberculin-negative recipients with the white cells from each tuberculin-positive donor. When cells from tuberculin-negative donors were used for sensitization, slight reactions were obtained in one-half of the recipients. When sites were prepared with cells

obtained from patients with sarcoidosis, no significant reactions were obtained. Similar studies were done on 6 subjects with sarcoidosis. In these recipients, tuberculin reactions were again consistently transferred with white cells from tuberculin-positive donors. Small to insignificant reactions were obtained in the sarcoid recipients with cells from tuberculin-negative donors. One tuberculin-positive subject received cells from a patient with sarcoidosis and a positive reaction was obtained. Thus, cells from patients with sarcoidosis did not inhibit the tuberculin reaction in known reactors, nor were they capable of sensitizing nonreactors. These findings are in conflict with the hypothesis that positive anergy is due to the presence of anticutins in sarcoid skin. Furthermore, the theory that patients with sarcoidosis have a masked hypersensitivity to tuberculin is also untenable in the light of these findings. It is suggested that tuberculin anergy in sarcoidosis is nonspecific and is due to involvement of the reticuloendothelial system by the disease. B. S.

Miscellaneous Allergies

Peripheral Neuropathy and Serous Meningitis Due to Horse Serum. Report of a Case With Failure to Respond to Cortisone. Walsh, J. W.: New England J. Med. **247**: 88, 1952.

A 27-year-old man was admitted to the hospital because of fever for six days and urticaria and pains in the neck and arms for twelve hours. Twelve days prior to admission, he had injured his finger on a wire fence and the traumatized area became infected. Six days after the injury, he was treated for this condition with intramuscular penicillin and tetanus antitoxin. About three hours after this therapy, he experienced right-sided chest pain, headache, weakness, and a slight rise in temperature. The next day he noted pain, redness, and itching at the site of the antitoxin injection and his temperature rose to 101° F. The infected area on his finger was incised. Despite antihistamine therapy the itching increased, and fever, sweating, and weakness persisted. He was hospitalized on the sixth day after the antitoxin injection, at which time he had generalized urticaria and pains in the outer aspect of both arms, radiating to the cervical spine, and pains in the thighs. There was some resistance to movement of the neck, but the Kernig and Brudzinski signs were negative. Motion at the right elbow was diminished. The biceps, triceps, and supinator reflexes were absent on the right and diminished on the left. Relief of pain was obtained temporarily with intravenous injections of procaine and with Demerol. However, definite signs of weakness of both shoulders were noted, especially on the right, and there was flattening of the right deltoid muscle. Flattening of the right nasolabial fold was also observed. Cerebrospinal fluid examination revealed a cell count of 133 lymphocytes, a trace of globulin, and a protein of 98.5 mg. per 100 c.c. The colloidal gold test gave a meningitic type of reaction. The heterophile antibody titer in the blood was 1:896, but heterophile agglutitins were not found in the spinal fluid. Despite large doses of Pyribenzamine, thiamine and vitamin B complex, the patient's condition became worse. Marked tenderness developed over the brachial plexuses, he could not move his right shoulder actively, and sensory changes occurred. Cortisone was then administered intramuscularly for ten days without any visible improvement. The pains disappeared about three weeks after admission to the hospital. The patient was discharged from the hospital four months later with marked atrophy of the deltoids, supraspinati, and infraspinati. When seen six months later, he had residual weakness and atrophy, especially on the left. He gave a history of having received tetanus antitoxin twelve years previously without ill effects. B. S.

Food Allergy With Special Reference to Corn and Refined Corn Derivatives. Bernton, H. S.: Ann. Int. Med. **36**: 177, 1952.

The various views on the incidence of hypersensitiveness to corn meal, cornstarch, corn oil, and corn syrup are reviewed. The author subjected 50 consecutive cases to an ingestion test with corn meal. Only 2 of the patients reacted positively. Further studies were carried out on one of the positive reactors, a 37-year-old woman with bronchial asthma. She gave a history of the induction of asthma, not only from the eating of corn, but also from the inhalation of steam arising from boiling corn. She also stated that the ingestion of cornstarch produced mild asthmatic symptoms. Intracutaneous tests with corn extract gave negative reactions. Corn meal, starch, and oil were applied to scratches on the skin and essentially negative reactions were observed. Ingestion tests were performed on a fasting stomach after the patient had abstained from corn products for four days. The patient did not know what food was being fed. The test foods consisted of corn meal mush, cornstarch pudding, arrowroot pudding, peanut oil, corn oil, and corn syrup. She developed asthma after the ingestion of the corn meal and cornstarch only. In another study with this patient, cornstarch was applied to the buccal mucosa. She responded to this with an asthmatic attack and also developed small blisters at the site of application of the starch. Passive transfer studies with this patient's serum failed to reveal reagins for corn, either by direct testing of the sensitized sites with corn extract or by feeding corn to the subjects. The author states that the dictum, "the treatment of corn allergy entails the elimination of all sources of corn," is open to question. B. S.

Thrombocytopenic Purpura Following Quinidine Therapy. Nussbaum, H. E., and Shulman, M. W.: Ann. Int. Med. **37**: 190, 1952.

A 65-year-old man who was treated with quinidine sulfate for auricular fibrillation began to complain of pain in his legs and feet about nine weeks after beginning therapy. His legs showed a petechial and purpuric eruption and showers of petechiae were noted over the rest of the body. He had had gingival bleeding for several days before the eruption was noted. The platelet count was 36,000 per cubic millimeter at this time. Seventeen days after discontinuation of the quinidine, the count was normal and clinical recovery had occurred. After recovery, a thrombopenic test was performed to establish quinidine as the causative agent. Two control platelet counts were done one hour apart. Then 0.2 Gm. of quinidine sulfate was given orally. In one hour, the platelet count dropped 50 per cent, and in four hours it was 90,000 as compared to the control level of 280,000. There were no manifestations of purpura or bleeding following the test dose. The rapid drop in the platelet count following the test dose suggests that the drug acts directly on the circulating platelets. B. S.

Anaphylaxis

A Comparison of the Effects of Anaphylactic Shock and of Chemical Histamine Releasers. Mongar, J. L., and Schild, H. O.: J. Physiol. **118**: 461, 1952.

Guinea pigs were sensitized to egg white by subcutaneous and intraperitoneal injections. Various tissues were removed from the animals three to six weeks later and washed with Tyrode's solution. The tissues were then suspended in Tyrode's solution and exposed to the specific antigen or to a chemical histamine liberator, such as D-tubocurarine or compound 48/80.

For the purpose of comparing the amount of release by these 2 types of substances, adjacent pieces of tissue of similar thickness and structure from the same animal were used. Strips of guinea pig ileum were employed to demonstrate the amount of histamine liberated from each tissue section. The amount of histamine released either by antigen or by chemical activators varied markedly in different tissues. The amount released was least in the intestine and greatest in the diaphragm, aorta, and uterus. The largest absolute quantity of histamine was liberated from the lungs.

There was a close parallelism between the percentage of histamine released in the anaphylactic reaction and by the 2 synthetic releasers. There was no correlation between the histamine content of the tissues and the amount of histamine released. Unsensitized tissues released no histamine following antigenic exposure, but, in these same specimens, compound 48/80 released quantities comparable to those of sensitized tissues. In all the tissues examined following a single ten-minute bath exposure, only a fraction of the total histamine contained in the tissue was released by the chemical releaser or by the antigen.

To determine the influence of the chemical releasers and the antigen upon each other, tissue was placed in solutions of one releaser until the rate of histamine release fell to a steady low value. The tissue was then exposed to solutions of a second releaser and the change in rate of histamine release was measured. In almost every instance, histamine liberators were able to release histamine following antigenic exposure. The synthetic drugs were so capable of releasing histamine in certain tissues that antigenic stimulation was not always effective in releasing histamine from tissue previously exposed to the chemical agents. It is suggested that the parallelism between histamine release by chemical liberators and by antigen might possibly be due to the formation, during an antigen-antibody reaction, of some degradation product of proteins which acts in much the same way as either 48/80 or D-tubocurarine. R. W.

Quantitative Study of Arthus Reaction and of Cutaneous Anaphylaxis Induced Passively in the Rat. Ovary, Z., and Bier, O. G.: Proc. Soc. Exper. Biol. & Med. **81**: 584, 1952.

Reverse Arthus reactions were produced in rats by injecting fixed amounts of egg albumin (Ea) or pneumococcal polysaccharide type III (SIII) intravenously followed immediately thereafter by an intracutaneous injection of fixed amounts of rabbit anti-Ea or anti-SIII antibody nitrogen. With both antigen antibody systems, the amount of antibody required for minimal reverse Arthus reactions was between 5 and 10 μg of antibody nitrogen. This value was essentially the same as that found by other workers in guinea pigs. Passive cutaneous anaphylaxis reactions were elicited in rats by the intracutaneous injection of fixed amounts of antibody followed four hours later by the intravenous injection of a mixture of antigen and a dye. The intensity of the reactions was measured by the size of the circular blue spots which developed at the sensitized sites. The amount of antibody necessary for the production of a minimal passive cutaneous anaphylaxis reaction was 0.5 to 1.0 μg of antibody nitrogen with both antigen antibody systems. About 100 to 200 times more antibody was required to produce similar reactions in the guinea pig. The difference in the minimal amounts of antibody required for passive cutaneous anaphylactic reactions, as contrasted with the similar amounts required for the passive reverse Arthus reaction in the rat and in the guinea

pig, indicates that the 2 reactions are basically different in their mechanisms. Studies with the direct Arthus reaction in the rat indicated that the severity of the reaction was directly related to the amount of antibody used in its production. This finding is in agreement with those previously reported in the guinea pig and rabbit. B. S.

Mechanism of Anaphylaxis in the Rabbit. Further Evidence Against Plasma Protease Mechanism. McIntire, F. C., Roth, L. W., and Sproull, M.: Proc. Soc. Exper. Biol. & Med. **81**: 691, 1952.

The addition of homologous antigen to sensitized rabbit blood in vitro produced a release of a large amount of histamine, but failed to activate a detectable amount of plasma protease. Conversely, the addition of streptokinase to sensitized rabbit blood markedly activated plasma protease, but failed to release significant amounts of histamine. Plasma protease levels in the blood of normal rabbits and in the blood of sensitized rabbits during severe anaphylaxis were compared and no significant difference could be found. When streptokinase was injected into normal rabbits, a marked elevation of the plasma protease level was produced in most cases, but anaphylactic symptoms were never elicited. Thus, the plasma protease theory of the mechanism of anaphylaxis in the rabbit appears untenable. B. S.

Pharmacology, Physiology, and Pathology

The Production of Antibody by Patients With Acute Viral Hepatitis. Eichman, P. L., Miller, R. W., and Havens, W. P., Jr.: J. Immunol. **70**: 21, 1953.

The capacity of patients with acute viral hepatitis to form antibodies was studied by measuring their immune response following stimulation with diphtheria toxoid. Anamnestic responses in Schick-negative patients were studied rather than primary responses in Schick-positive patients because the production of antibodies in the former group was greater and followed a more uniform pattern. Thirty-four Schick-negative subjects with hepatitis were injected intramuscularly with 50 Lf (0.14 mg.) of highly purified diphtheria toxoid between the fourth and twenty-first day of their illness. Blood was drawn before Schick tests were done and also between the seventh and forty-second day following administration of the toxoid. Antibody levels were measured by means of titrations performed in the skin of rabbits. Thirty-one control subjects who were either in apparent good health or suffering from minor orthopedic disorders were similarly infected with diphtheria toxoid after their Schick negativity had been established. It was found that there was a definite similarity between the test and control groups in the qualitative and quantitative pattern of production of diphtheria antitoxin. The patients with hepatitis, however, had increased amounts of antibody in the blood specimens taken before the Schick tests were done. There was no noticeable relationship between the capacity to form antibody and the age of the patient, severity of the hepatitis, or degree of hyperbilirubinemia. It is suggested that the higher initial antibody levels in the patients with acute hepatitis may have been due to an anamnestic reaction from the disease itself. The authors conclude, however, that the hepatitis, per se, had little if any effect on the quantitative or qualitative ability to form antitoxin following stimulation with diphtheria toxoid. R. W.

Antibody Production in Inbred Strains of Mice. Fink, M. A., and Quinn, V. A.: J. Immunol. **70**: 61, 1953.

Five strains of mice were continuously inbred for more than 20 generations in order to insure genetic uniformity. Animals of the same age among the different strains were injected intra-abdominally or intramuscularly with egg white or pneumococcus polysaccharide. There was a definite genetic variation in antibody response which was independent of the route of administration or chemical composition of the antigen. However, consistently higher titers were obtained when the animals were sensitized by an intra-abdominal rather than by an intramuscular injection. Egg white antigen was found to produce a greater antibody response than pneumococcus polysaccharide antigen. It was also found that increasing the altitude at which the experiments were performed caused a slight rise in the immune response. Sex did not influence the antibody response obtained. Younger mice produced much less antibody than the more mature animals. The authors conclude that there is a genetic difference in the ability of mice to produce antibodies. They also suggest that "the mouse does not form antibody to antigens of different chemical constitution in an identical manner." R. W.

Antibody Formation in Vitro by Haemopoietic Organs After Subcutaneous and Intravenous Immunization. Thorbecke, G. J., and Keuning, F. J.: J. Immunol. **70**: 129, 1953.

Young adult rabbits were immunized either subcutaneously or intravenously with paratyphoid B vaccine. Three days after the last of a series of 3 immunizing injections had been administered, the animals were sacrificed and the organs were removed for study. Tissue taken from each organ was divided into 2 equal parts. One-half was immediately macerated, and extract was made and the antibody titer was measured. The other portion was incubated for twenty-four hours at 37° C. and then similarly prepared for immunologic evaluation. An increase in the antibody titer of the incubated tissue over the titer found in the tissue studied immediately was interpreted as evidence of production of antibody. The liver and thymus produced practically no antibodies. Considerable quantities of antibodies, however, were formed by the bone marrow, lymph nodes, and spleen. The red splenic pulp was by far the largest producer of antibodies, regardless of the route employed for the immunizing injections. When the injection was given subcutaneously, the lymph node on the injected side always showed a high antibody titer. When sensitization was carried out by the intravenous route, the lymph node titer was always low. Increased numbers of plasma cells were observed microscopically in the tissues (spleen and bone marrow) producing high titers of antibody. It appeared that, when immunization was produced by the technique previously described, the greatest of antibodies took place in the red pulp of the spleen. R. W.

Degenerative Forms of Eosinophilic Leukocytes in Lymphoid Organs. Fruhman, G. J., and Gordon, A. S.: Proc. Soc. Exper. Biol. & Med. **82**: 162, 1953.

In a study of touch prints and histologic sections of ileocecal lymph nodes and other lymphoid organs of normal rats, eosinophiles were found in varying stages of disintegration. These degenerative forms were classified as eosinorrhexicytes and eosinomeres. The former were small cells with changes in the nuclear material, while the eosinomeres were anuclear and consisted of eosinophilic granules varying in size and amount. Approximately 35 per cent of

the eosinophilic forms in the ileocecal lymph nodes of rats were found to be degenerative. When the animals were exposed for six hours to a low barometric pressure the percentage of degenerative forms doubled and a peripheral eosinopenia developed. It remains to be determined whether active destruction of the eosinophile occurs within the lymphoid organs or whether the degenerative forms are merely being filtered out at this site. M. COHEN.

Effect of Some Steroids and of Corticotropin (ACTH) on Cellular Activity. Menkin, V.: Proc. Soc. Exper. Biol. & Med. **82**: 189, 1953.

Two factors capable of increasing capillary permeability have been found in inflammatory exudates, namely, leukotaxine and exudin. Leukotaxine is present early in the inflammatory process when the exudate is alkaline and has the property of attracting leukocytes. Cortisone and compound F suppress the action of leukotaxine, while ACTH fails to affect it. Exudin is found later in the inflammatory reaction, when the exudate has become acid. Its action is repressed by ACTH, but is not influenced by cortisone or compound F. In the present study, the author sought to account for the suppressive action of these hormones on the inflammatory reaction.

It has been shown that the cellular division of fertilized Arbacia (sea urchin) ova may be employed as an index of normal cellular activity. When the ova are adversely affected by a substance, the incidence of cleavage is reduced and the pattern of cell division becomes atypical. Cortisone, compound F, desoxycorticosterone, and ACTH were found to inhibit cleavage of these ova. On the basis of these observations, it is suggested that these hormones exert their suppressing effect on inflammation by directly reducing cellular activity. This results in decreased formation of leukotaxine and of other biological substances which influence the inflammatory reaction.

M. COHEN.

The Localization of Homologous Plasma Proteins in the Tissues of Young Human Beings as Demonstrated With Fluorescent Antibodies. Gitlin, D., Landing, B. H., and Whipple, A.: J. Exper. Med. **97**: 163, 1953.

Specific antisera were prepared by injecting purified fractions of human plasma into rabbits. The resulting antibodies were conjugated with fluorescein isocyanate and used as histochemical "stains" on sections of human tissue obtained by biopsy or autopsy and examined under the fluorescence microscope. The serum fractions used were human plasma albumin, γ-globulin, β-lipoprotein, B_1-metal-combining globulin, and fibrinogen. "For the most part, antigen was detected with anti-β-lipoprotein (anti-B) in the nuclei of all cell types examined, while with anti-fibrinogen (anti-F), antigen was restricted largely to the lymphatic and vascular channels, the connective tissues, and the interstitial spaces. Antigen revealed with anti-γ-globulin (anti-G), anti-albumin (anti-A), and anti-β_1-metal-combining globulin (anti-FE) was localized in lymphatics and blood vessels, connective tissues and interstitial spaces, and in the nuclei of many cells. Antigen in connective tissue appeared to occur between collagen fibers, as if in the matrix or fine spaces rather than in the fibers themselves." The specific localization of the above-named serum fractions is presented for liver, kidney, spleen, lymph nodes, thymus, lung, trachea thyroid, adrenal, heart, skeletal muscle, tongue gastrointestinal tract, pancreas, brain, and skin. It is suggested that the intranuclear presence of γ-globulin may be adequate to protect a cell from those infectious agents which require cell nuclei for their survival. Thus tissue

immunity may be present in the absence of detectable circulating antibody. Relatively large amounts of plasma proteins were found in connective tissue and to a lesser extent in cells. This may represent a reservoir for the maintenance of constant plasma protein levels. K.

The Occurrence of Focal Tuberculoid Lesions in Experimental Serum Sickness. Rich, A. R.: Bull. Johns Hopkins Hosp. **91**: 109, 1952.

Serum sickness was induced in rabbits by a single intravenous injection of 10 c.c. of sterile horse serum per kilogram of body weight. This dose was used in order to permit some antigen to remain in the circulation after antibody formation had taken place. The animals were sacrificed from the twelfth to the sixteenth day following sensitization. Obvious tuberculoid lesions were found only in the spleen. Some of the lesions were strictly perivascular, while other tuberculoid responses were found adjacent to foci of necrosis in the arterial wall. There was a marked variation in the extent of tuberculoid responses found in the different animals. There was no correlation between the development or degree of anaphylactic tuberculoid lesions and the cardiovascular or renal lesions of hypersensitivity. There was no relationship between the development of tuberculoid foci and antibody titers (antigen-dilution precipitin tests) performed two weeks following immunization. There was also no correlation between skin sensitization as measured by intracutaneous reactions and tuberculoid responses. In animals where the sensitizing dose was limited to a single intracorneal injection of 0.025 c.c. of horse serum, skin test reactivity and antibody titers two weeks later were strongly active, but no tuberculoid foci were found in the microscopic sections of the spleens. The incidence of tuberculoid responses appeared to be related to the quantity of the sensitizing antigen injected.

Fifty-seven animals were similarly sensitized with horse serum but were given either 10 mg. of ACTH or 7.5 mg. of cortisone daily until they were sacrificed. Those treated with ACTH had slightly less extensive tuberculoid lesions while the animals treated with cortisone had a significantly lower incidence of tuberculoid responses. It is suggested that the study of tuberculoid lesions in hypersensitivity may help to solve the pathogenesis of tuberculoid lesions in infection. R. W.

The Mechanism of Dust Clearance From the Lung. A Theory. Gross, P.: Am. J. Clin. Path. **23**: 116, 1953.

Lung sections from autopsies on 87 adults were examined. In 43 cases, moderate to large numbers of alveolar macrophages were found, without other inflammatory cells. The magrophages were localized near atelectatic areas, especially when the atelectasis was only partial or when the alveoli were edematous. Their presence was also sharply focalized "in the vicinity of bronchi, vessels and scars, or within alveoli with thickened walls." The cells were pigmented with carbon in many instances; in cases of chronic heart failure, the pigment was largely hemosiderin. The author suggests interference with the respiratory excursions of the alveoli leads to failure to move the thin film of mucus covering the alveolar surface with consequent stasis of dust particles and of macrophages in this film. Changes of viscosity of the mucus film during the respiratory cycle probably also favor movement of mucus and its contained particulate matter toward the ciliated areas of the bronchioles. K.

Miscellaneous

Allergic Purpura, Including Purpura Due to Foods, Drugs, and Infections. Ackroyd, J. F.: Am. J. Med. **14**: 605, 1953.

Allergic purpura is divided into two types: (1) that which is associated with an erythematous exanthem and also with joint and visceral symptoms, the Henoch-Schönlein syndrome; and (2) true purpura, which is never associated with an erythematous exanthem.

Henoch-Schönlein disease, although regarded generally as an allergic disturbance, is due to food sensitivity in only a small percentage of cases. In most cases, the etiologic factor cannot be established. The purpuric lesions in Henoch-Schönlein syndrome are characteristically distributed over the extensor surfaces of the extremities, the buttocks, and the lower back, but may be present on any portion of the body. They occasionally are hemorrhagic and resemble the lesions of erythema exudativum multiforme. Many authors believe the two conditions to be identical. Males are more commonly affected than females, and children and adolescents more commonly than adults. Recurrences are frequent and may continue for months and even years. Hematuria is common, but epistaxis and bleeding from the gums are not seen as frequently as in other types of purpura. Nephritis is the most common serious complication and is indistinguishable from Type I nephritis of Ellis. Increased capillary fragility and eosinophilia can be demonstrated only in certain cases and there is usually no disturbance in the platelet count, the bleeding time, the coagulation time, or clot retraction. Diagnosis is made by the association of purpuric erythematous skin lesions with visceral or arthritic symptoms in a patient with a normal platelet count. Differential diagnosis must exclude acute abdominal conditions, but must take into consideration that Henoch-Schönlein syndrome is an occasional cause of perforation of the gut or of intestinal obstruction, usually as a result of intussusception. A careful food history, trial diets, and elimination diets may be of assistance in identifying an offending food. Skin tests are seldom helpful. Because of the frequency of nephritic complications, bed rest is of paramount importance in the treatment of the condition.

Some authors are of the opinion that the Henoch-Schönlein syndrome is a manifestation of bacterial allergy, particularly to the streptococcus and the tubercle bacillus, but no real proof of this etiology exists.

The second type of allergic purpura is that which occurs in the absence of any inflammatory or exanthematous reaction and is referred to as true purpura. It may be due to an abnormal response to an infection or to a drug, but is rarely if ever due to food sensitivity. Purpura may occur at the height of an infection or during convalescence, and its appearance is not dependent upon the severity of the primary disease. The platelet count may be normal or reduced in varying degrees. Both thrombocytopenic and nonthrombocytopenic purpura have been reported after all types of infections.

Purpura due to drugs has been reported following the use of a large number of substances. A drug may have been taken over a period of a few days, weeks, or even years without untoward results. Then, after a single dose, purpura may suddenly appear. Thrombocytopenia is usually the rule, but the nonthrombocytopenic variety does occur occasionally. Although the cases are usually of a mild character, persistent use of a drug in the face of sensitivity may result in a fatal reaction. In the case of drugs such as bismuth or gold, which are slowly liberated from the injection site, sensitivity may first become apparent months after the drug has been discontinued. Sensitivity to any drug may persist for years after discontinuance of its use.

In drug sensitivities the megakaryocytes have been reported as reduced, unchanged, or increased in number, and morphologically they may or may not be affected. This is in contrast to the many reports of gross changes in the megakaryocytes in purpura due to infections. It has been shown that thrombocytopenia due to drugs results from the action of an antibody which, in the presence of the drug, causes platelet lysis, whereas in the purpura due to infection the action is mainly on the megakaryocytes. However, in both conditions the platelets and the megakaryocytes must be affected and, since platelets are derived from the cytoplasm of the megakaryocytes, the same conditions which influence the platelet count probably affect the megakaryocytes. Evidence exists which indicates that platelets and capillary endothelium are antigenically related and that an antibody which can injure one can also injure the other in varying degrees.

Purpura from whatever cause is characterized by a tendency to bleed into the skin or mucous membranes. An increase in capillary fragility is of primary importance in the production of purpura since hemorrhage cannot occur in the face of an intact vascular endothelium. The existence of a vascular lesion in purpura can usually be demonstrated by one of the capillary fragility tests but, as a rule, no vascular defect can be detected histologically. Nevertheless, much evidence exists which indicates that the basic lesion is in the endothelium. Although thrombocytopenia is not the important factor in purpura, it may play a part in increasing the hemorrhagic tendency. F.

Studies on the Generalized Shwartzman Reaction. II. The Production of Bilateral Cortical Necrosis of the Kidneys by a Single Injection of Bacterial Toxin in Rabbits Previously Treated With Thorotrast or Trypan Blue. Good, R. A., and Thomas, L.: J. Exper. Med. **96**: 625, 1952.

Inasmuch as cortisone-treated rabbits become as susceptible to a single dose of meningococcal toxin as animals which are exposed to the second of 2 doses of this toxin, it became of interest to determine whether cortisone or the initial toxin dose might act by interference with the removal or fixation of the subsequently administered circulating toxin. To test the possibility that the reticuloendothelial system might be involved, Thorotrast or trypan blue was used as a blocking agent. Rabbits pretreated with these agents were injected with a single intravenous dose of *S. marcescens* toxin. They developed bilateral cortical necrosis of the kidneys in contrast to normal rabbits which suffered no ill effects. The renal cortical necrosis was typical of that seen in the generalized Shwartzman reaction. Hemorrhagic necrosis was also observed in lung, spleen, liver, thymus, and gastrointestinal tract. The enhancement of the Shwartzman reaction by Thorotrast did not appear when Thorotrast was administered six, eighteen, or twenty-four hours after the injection of toxin. Simultaneous injection of toxin and Thorotrast still resulted in the regular appearance of renal necrosis as it did when Thorotrast was injected three to twelve hours prior to the toxin. When Thorotrast was given twenty-four to forty-eight hours prior to toxin, renal lesions appeared in a few animals. Trypan blue was less effective than Thorotrast, larger doses of toxin being required. Again there was no effect when this dye was administered after the toxin.

The local Shwartzman reaction was also elicitable with a single intracutaneous injection of toxin in trypan blue or Thorotrast blockaded animals. These animals furthermore developed bilateral cortical necrosis of the kidneys as cortisone-treated rabbits do following the injection of toxin, and in contrast to the results in animals treated only with toxin (intracutaneous eliciting dose). Again, if the blocking agent was given after the dose of toxin no

lesions appeared. It was noted that animals injected with trypan blue eighteen hours after an intracutaneous injection of toxin showed deep blue staining of the skin except at the site of toxin injection. This finding could not be explained. To test whether absorption of toxin from the skin was enhanced by Thorotrast, titrations of intravenously and intracutaneously administered meningococcal toxin were carried out in Thorotrast pretreated rabbits. Bilateral cortical necrosis of the kidneys occurred in animals receiving dilutions of toxin as high as 1:2,560 intracutaneously. When the toxin was injected by vein, a 1:110,000 dilution but not a 1:20,000 dilution produced these lesions. It is concluded that "much of the intradermally injected toxin was absorbed from the skin in the thorotrast treated animals." In normal rabbits very large doses of toxin may be injected intracutaneously without affecting other skin areas previously prepared with toxin, indicating poor absorption. When other colloidal or particulate solutions (rabbit liver suspension, vegetable starch suspension, rabbit liver glycogen, human serum, egg albumin in egg-sensitized rabbits, polyvinylpyrrolidone) were injected prior to the intravenous injection of toxin, the rabbits were not made more susceptible to toxin.

Leukocyte counts were followed in rabbits pretreated with Thorotrast and given the smallest dose of toxin known to produce consistent renal lesions (1:10,000). A moderate leukocytosis followed the Thorotrast injection. Two hours after toxin administration, leukopenia was noted in both Thorotrast-treated and control animals, the degree being greater in the Thorotrast-treated animals. The leukocyte levels became normal within two to four hours of the toxin injection, remaining normal in the controls and becoming markedly elevated in Thorotrast-treated rabbits. Nitrogen mustard in leukopenia-producing doses circumvented the appearance both of the renal lesion and the skin lesions in Thorotrast-toxin-treated animals. It was noted that rabbits treated with Thorotrast required only a 1:10,000 dilution of toxin to precipitate renal necrosis and death, whereas animals prepared with cortisone or with intravenous injection of toxin required a dilution of 1:20 or 1:40. The results are "consistent with the concept that thorotrast acts by interfering with a mechanism which is normally responsible for the removal or detoxification of bacterial toxin." K.

Antigen Retention in the Rabbit. Stevens, M.: J. Exper. Med. **97**: 247, 1953.

Radioactive iodinated bovine gamma globulin was injected into the ear veins of rabbits. Blood was taken from the opposite ear after fifteen minutes and then daily for fourteen days. Radioactivity counts were carried out on the specimens removed and corrected for radioactive decay. One of two groups of rabbits was subjected to enough x-ray irradiation to inhibit antibody formation. Three or four months later, both groups were reinjected with I^{131} bovine gamma globulin. The control group showed an accelerated rate of loss of radioactivity as compared to the response to the primary injection in this group. The irradiated group behaved, on reinjection of antigen, like the control group on primary injection. It is argued that, since rabbits recover from x-ray in less than two months, if antigen were retained past this point, they should have then formed antibody and exhibited an anamnestic response to the second antigen injection. Since they did not exhibit the anamnestic response, it is suggested that the antigen does not persist beyond this time. K.

Role of the Vitamins in Antibody Production. Axelrod, A. E.: Metabolism **2**: 1, 1953.

Severe impairment of antibody response was observed in rats deprived of pantothenic acid, pyridoxine, and pteroylglutamic acid. Moderate impair-

ment was associated with riboflavin, thiamin, biotin, vitamin A, and niacin-tryptophane deficiencies. The withdrawal of vitamin B_{12} and of vitamin D had no effect on antibody response. Control experiments eliminated the possibility that the depression of antibody titer was due merely to inanition rather than to the vitamin deficiencies. It was further shown that the low antibody levels were due not to an increased rate of destruction of antibodies but rather to impaired synthesis.

The author concluded that certain B vitamins, particularly pyridoxine, pantothenic acid, and pteroylglutamic acid, play a significant role in antibody synthesis. H.

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